SOME CLINICAL REMARKS ON

PNEUMONIA.

BY

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PHYSICIAN, NORTH LONDON HOSPITAL FOR CONSUMPTION AND DISEASES OF THE CHEST.

Reprinted from THE LANCET April 4, 1896

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SOME CLINICAL REMARKS ON PNEUMONIA.[1]

THE latter part of the present century has so advanced our knowledge of the influence of bacteria on most pathological processes that a study of their relation to the more important pulmonary diseases claims special attention. The various parts taken by micrococci and by certain distinct kinds of bacilli and how far they share in the initiatory and sequential phenomena of the differing forms of inflammation of the lung have to be considered. Not only has the germ theory of disease given increased interest to pathological investigation and to experimental research, but it has resulted in larger and clearer views of etiology and has important bearings upon clinical medicine both in prognosis and therapeutics. In the latter sense I do not here refer to the attempts at treatment by bactericidal drugs ; this is now in the actively empiric stage, but it is one thing to kill bacteria in a test-tube and another to destroy them in the body. In the study of disease causation by the light of our knowledge of morbific organisms two factors demand attention—the seed and the soil; not only must the micro-organism be studied, but the conditions under which it will flourish. This is well recognised by the bacteriologist in preparing his culture media, and to the clinical observer and practical physician the influence of the soil on the life and virulence of the disease-germ are of the greatest importance. The "personal" equation in medicine has received a new interest and an increased importance from the discovery of the association of bacteria with certain diseases. The influence of the "personal equation" in disease is no new discovery, but we may note in the present day a greater tendency than was formerly the rule to treat individuals rather than diseases. In pneumonia the influence of the personal factor is well seen. Inflammation of the lung in a healthy child is a very different matter from the same accident occurring in an adult of impaired or shattered constitution, even though the initial lesion were of less extent or intensity. It is this individuality of each case of pneumonia which, whilst it renders the disease exceptionally interesting to the clinician, makes the management of each case a matter of peculiar anxiety. I propose to go through my case-book and touch upon some points of interest which my experience has furnished.

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[1] A paper read before the Harveian Society on Feb. 20th, 1896.

Etiology.—It is now almost universally agreed that acute lobar pneumonia must be considered as a general, not merely a local, disease. And although there are difficulties in the way of fully accepting this view there are many features of the illness which give it strong support. The discovery by Friedländer and Fraenkel of the micro-organisms with which their names are now associated seemed to furnish the proof of the general nature of the illness. We must, however, remember that similar or identical organisms are associated with other diseases besides pneumonia and are to be found in the saliva in health, and probably exist in the lungs and other organs without producing ill-effects. When some local change in the tissues furnishes a suitable soil these micro-organisms develop rapidly and produce inflammation, whilst the toxic substances formed by them give rise to general symptoms. These local changes may be caused in various ways—for example, by traumatism or by chill—and they are more readily induced in organs of lowered vitality, whether this is permanent or temporary. Thus we find alcoholism predisposes to pneumonia, and that inflammation of the lung is a common accident in many illnesses where the general vitality of the tissues is impaired—e.g., typhoid fever and influenza. So, too, the fatigue consequent upon excessive exertion renders a chill more dangerous ; the overdone competitor in a long distance bicycle ride finishing in a cold drizzle is more likely to get pneumonia than the spectator who has stood shivering to watch the result. It is a fact, sometimes imperfectly recognised, that over-fatigue is an important factor in the production of pneumonia. I have seen more than one case of pneumonia following a chill during the latter part of a long bicycle ride. The changes in the lung tissue which favour inflammation are probably the result of nerve influence which may originate at a distance from the affected organ. We know that in animals inflammation in the lung will result from injury to the vagus. The influence of chill to the surface of the body probably affects the lung by reflex nerve action. Such a result is perhaps most likely to ensue with exhausted nerve control, and pneumonia from chill is thus more prone to happen during fatigue. The pneumonia of alcoholism has been considered to be a trophic neurosis originating at the vagus centres, and possibly the poisons of enteric fever and influenza may act in a similar manner. On the other hand, the lung tissue may be directly damaged by local irritation. This may be mechanical, as from a blow, fractured ribs, or inhaled mineral particles, or the local irritation may in some cases be due to the toxic effects of micro-organisms. It may be that even local irritation produces its effects by reflex nerve action. These considerations may help us to understand the occurrence of pneumonia under such widely different conditions as traumatism and chill, or as a secondary result of disease.

Just as fatigue increases the risk from chill, so may the debility after an illness render a chill more dangerous. In the following case we have a good example of the effect of measles in predisposing to pneumonia from chill. A man aged about thirty-five years had an attack of measles, which went through its course without any noteworthy characters until the patient was sufficiently well to sit up in his bedroom for an hour or two during the day. Being so well he sent the nurse out for a walk and in her absence he took a bath, and then, without completely dressing, he threw open his window, although it was in March, and began to arrange some papers. That evening he had a rigor, and the temperature, which had been normal for some days, ran up. He had a sharp attack of pneumonia. The readiness with which convalescents from measles suffer—and often suffer severely—from chill, though well known to medical men, is imperfectly appreciated by others, and the large mortality after measles is due to the preventible complications and sequelæ. The case I have just mentioned is one of idiopathic pneumonia following after measles and is quite different from the pneumonia which occurs in measles as a part of the disease, coming on before the original fever has disappeared. The lungs are always affected to some extent in measles (as a part of the disease) and are liable to remain for a time in a condition which renders them a suitable soil for the development of any micro-organisms which may be present in these organs already or which may get to them from outside. Thus we find measles often an antecedent to pulmonary tuberculosis.

I have seen several cases of pneumonia associated with enteric fever and some of them have had this point of special interest—Was the pneumonia present the substantive disease and the actual cause of the illness or a complication not infrequent of enteric fever? The recognition of the signs of pneumonia has, in some cases which I have seen, been taken to explain the illness and so misled as to the true nature of the case. Whenever in obscure cases signs of pneumonia are detected it is important to consider whether the condition of the lung will sufficiently explain the course of the illness as well as the existing symptoms. As a good instance of the importance of this I may refer to a case I saw recently in consultation. I was asked to see a boy aged about eleven or twelve years because pneumonia had been diagnosed. On examination there was no doubt as to the presence of pneumonic patches in both lungs, especially the right, but the lung trouble did not seem to me sufficiently intense to explain the serious condition in which the boy obviously was at that time. Further examination disclosed pericarditis with effusion. Pericarditis may complicate pneumonia, but here on further inquiry I was led to a diagnosis of acute rheumatism, which the further course of the illness seemed to justify. The boy recovered, but with an endocardial murmur, which has now, after three months, almost disappeared.

The pneumonia of influenza presents some peculiar characteristics. The sputa may be rusty and the symptoms severe, but the physical signs are often obscure and the course of the disease unexpected. One marked peculiarity which I have noted is the tendency of the pneumonia to appear in some distant part of the lung or on the opposite side when resolving at the original seat Here is a characteristic case. A young man about twenty-five years of age was seen in consultation in the country. On the right side of the chest the percussion note was "tubular" (high-pitched and sharp, with diminished resonance, but not dull) from the apex to the fourth rib, with tubular breathing. The rest of the right front was clear except for a few crepitations. The right back was dull almost all over. The left side was clear. Expectoration was very tenacious and had a slight brick-red colour, not typically pneumonic. The medical history showed that the pneumonia commenced at the right base, and as that resolved the apex became affected with another rise of temperature. This is quite different from what is found in typical acute pneumonia.

With reference to the influence of malaria on pneumonia, in my experience at Suakin in 1885 malaria was not a frequent cause of illness, except in those cases which have been called "typho-malarial," and which I believe to be dependent upon malarial poisoning. In these cases, as well as in some of the cases of enteric fever there, pneumonia occurred as a complication. A malarial pneumonia has been described, but Osler, with his large experience of malaria, says he has never yet met with a case of pneumonia which seemed to be in any way connected with paludism. The debility which is induced by a malarial attack may, however, predispose to pneumonia, as in the following case. A man aged thirty-three years arrived in England in April from the West Indies, where he had been for about five months, and had been eight weeks seriously ill with malarial fever. I saw him in consultation the day after he landed and found pneumonia affecting the right apex. When I saw him again, three weeks later, the chest was clear, the pneumonia having completely resolved. Tubercle had been suspected as the cause of the lung mischief, but no bacilli were found in the sputa, and the complete resolution within three weeks is in itself almost sufficient to confirm my opinion that the pneumonia was not due to tuberculosis. The patient recovered and still keeps well after nearly three years. Whether malarial fever predisposes to pneumonia or not, the cachexia left after severe malarial poisoning adds to the danger of a subsequent pneumonia. Some years ago I attended a man aged thirty-nine years during an acute attack of pneumonia, from which he died on the tenth day. The patient had served in the marines and had suffered from malaria. The illness with the pneumonic attack was very acute, the temperature was high, with delirium from the fifth day. In this case I believe the previous malarial poisoning had left him without power to resist any severe illness, for his age and habits were not unfavourable to recovery.

The foregoing remarks will emphasise the importance of the "soil," in addition to the causative bacteria, as a factor in pneumonia. There is this analogy between pneumonia and tuberculosis: in both a micro-organism is the essential cause, but the germ is harmless unless the soil is favourable to its development. The difference between the two diseases depends upon the different characteristics of the causative micro-organisms. The pneumococcus or pneumo-bacillus, developing rapidly, quickly produces its full effects, and sufficient toxines are formed to cause an acute illness and usually to poison the germs before they can become widely distributed. Thus we have a sharp but short illness usually terminating by crisis. The tubercle bacillus also produces a pneumonia, but develops slowly ; the consequent small total output of toxines in the early disease leaves the blood-stream non-poisonous to the bacilli, and they are carried by it to other parts, where they set up fresh centres of disease. In pneumonia, as in tuberculosis, the various predisposing causes, such as cold, exhaustion, or debility, render the individual susceptible ; the character of the "tissue soil" (to quote Osler[2]) has been so changed that the specific germ of either can grow and produce its ultimate effects.

Course and symptoms of pneumonia.—There is a very interesting point on which opinions are not unanimous—namely, whether there are cases which justify a diagnosis of acute congestion of the lung. The French consider active congestion of the lung to be a distinct disease (maladie de Woillez), but most authorities in this country consider the cases so described to be cases of pneumonia. Cases undoubtedly do occur, commencing suddenly with the symptoms which mark an attack of pneumonia, which either terminate fatally before the lungs become consolidated or which resolve without going through the ordinary course of pneumonia. Such cases may be termed "abortive pneumonia," and these are the cases to which the term "acute congestion of the lung" is sometimes applied. I select two of my cases bearing upon this point—one in a child, the other in an adult. A little girl aged four and a half years, who was said to have had congestion of the *left* lung the previous winter, was taken ill rather suddenly with feverish symptoms and slight cough. I found the temperature considerably raised and the base of the *right* lung dull. A mustard poultice was applied, and the next day the dulness had cleared and the temperature went down in two days. In the other case a woman past middle age, having just got over an illness of a few days' duration (supposed to be influenza), was found to have a return of fever with signs suggestive of pneumonia. When I saw her the next day the temperature was 101° F., and there was diminished resonance over the right base up to the angle of the scapula, with weakened breath sounds and fine crackling over the affected area. A mustard leaf was applied. I again saw her three days later. The temperature was down and the patient felt well. The right base was resonant, the breath sounds still somewhat weaker than on the left, but there were no crepitations. This was hardly a hypostatic congestion, for it was accompanied with a rise in temperature and the conditions which induce hypostatic congestion were absent. These two cases, though presenting different clinical phenomena, have this important characteristic in common—viz., that in both the febrile illness was short and the consolidation of the lung was rapidly absorbed, whereas the consolidation of pneumonia usually takes some time to resolve after the febrile illness is over. In this sense they may both be described as "abortive" pneumonia.

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[2] The Principles and Practice of Medicine, second edition, p. 547.

The pathological conditions found in pneumonia may be referred to in explanation of such cases. These, as given by Dr. Douglas Powell, consist "first of an acute hyperæmia of the affected lung resulting in a fibrino-corpuscular exudation into the alveoli, sometimes including the smaller bronchioles and forming a film upon the pleural surface. This exudation, secondly, coagulating *in situ*, fixes the lung in a state of immovable expansion more densely solidified than it could be by any artificial injection with coagulable fluid."[3] The facility with which these exudations may be absorbed varies with the nature of the effusion and of the corpuscular elements and the degree of coagulation obtained. In the cases of which I have just been speaking the fluid exudation is presumably absorbed before coagulation takes place. We have thus only the first stage of the pneumonic process.

It is unnecessary here to describe the symptoms and course of an ordinary case of pneumonia, but I propose to mention one or two peculiarities or divergencies from the normal course which have come under my notice. We sometimes find that in cases of acute pneumonia the fever subsides gradually instead of by crisis, and it has seemed to me that this is more common in hospital practice than in cases seen at home. Possibly removal of the patient may have something to do with this. If a patient is taken to hospital when the disease is well established—say from the third day onwards—the febrile period becomes somewhat prolonged and the crisis is delayed or wanting. When consolidation of the lung has occurred resolution necessarily takes time, and in the most favourable cases is not complete until some days after the temperature has fallen. We meet with cases, however, in which this resolution is delayed for a considerable period, it may be several weeks or even months. I have notes of a case of a girl aged twelve years in whom resolution was not complete for nearly two months, but it may be delayed longer than this. Such cases may get absolutely well, as this girl did, the lung eventually clearing up completely. In other cases where resolution is delayed we find some further mischief, either as the cause or a result of the delay; for example, tubercle or pleuritic effusion or an abscess may form in the lung. I have in a previous paper[4] before the Harveian Society drawn attention to the risk of tuberculosis after pneumonia, especially in those cases where resolution has been long delayed. The pneumonic process nearly always leaves the lung in a condition favourable to the growth of germs which may subsequently reach the organ ; thus we find that, unlike what is the rule in a general disease, one attack renders the person more liable to suffer again from pneumonia and predisposes also to phthisis. Some years ago I had a case of gangrene of the lung following pneumonia,[5] and I have lately had an interesting case of pneumonia affecting the upper lobe of the left lung in which a pyo-thorax developed which was also localised in the upper portion of the left chest. The patient, a boy ten years of age, was one of three inmates of a school who developed pneumonia. In due course the crisis occurred in all, but whereas two of the boys had an uninterrupted convalescence the temperature of the third, after being down for a day or two, ran up again and showed the large diurnal range of septic fever. The left chest was immovable in respiration and quite dull on percussion. The boy had a tuberculous family history, and I took him into hospital to have him under observation. Being satisfied of the presence of fluid in the pleural cavity I got Mr. Watson Cheyne to open the chest. A large quantity of offensive pus was evacuated and the boy made a good recovery. I have had several cases of empyema following pneumonia, but this case of empyema localised at the apex is exceptional.

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[3] Dr. Douglas Powell: Diseases of the Lungs, fourth edition, p. 221.

The case just quoted leads one on to refer to cases of apex pneumonia. Pneumonia affecting the apex of the lung is not necessarily more severe or more dangerous than inflammation occurring at the base. Cases of apex pneumonia are sufficiently common to make it unnecessary for me to quote instances here. One important point of interest in these cases is the difficulty of distinguishing between a simple pneumonia in this situation and tuberculosis. The apex of the lung being the most frequent seat of commencing tuberculosis we may be led to diagnose phthisis when we get consolidation of the apex without fully considering the history and physical signs. It is important also to remember that pneumonia may be induced by a patch of tubercle, the presence of which is not recognisable until the surrounding pneumonia subsides. I have elsewhere referred to cases illustrating this and other points in reference to the connexion between pneumonia and tuberculosis.[6]

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[4] Medical Press and Circular. April 20th 1892.
[5] THE LANCET, July 20th, 1889

With regard to the sputum in pneumonia, viscidity is perhaps a more general characteristic than the rusty colour. In catarrhal pneumonia the rusty colour is not infrequently absent, and it may be so in lobar pneumonia. On the other hand, I have seen the characteristic rusty sputum when careful examination failed to detect any dulness; here the pneumonic patch was probably deep-seated. The prune-juice expectoration, which is often a sign of danger, is not always so. The change from marked viscidity to a more liquid as well as darker sputum is more significant of danger than the deepened colour alone.

Diagnosis.—Theoretically the signs of pneumonia are sufficiently characteristic to be recognised easily and with certainty, but practically we find ourselves often obliged to weigh carefully all the evidences obtainable before we can give an opinion. I have mentioned the difficulty occasionally met with in deciding between tuberculosis and pneumonia, and I have on more than one occasion found a pleural effusion where pneumonia has been diagnosed. Pleurisy may follow pneumonia, and when the signs of pneumonia have been correctly recognised in the first place dulness on percussion at subsequent examination has been interpreted as indicating non-resolution when it was in reality caused by fluid. Careful auscultation should be practised throughout the course of a case to avoid such an error, and repeated testing of the vocal fremitus must not be omitted; but even with this care the mistake is not always avoidable. The dulness of hypostatic congestion or of œdema from heart or kidney disease and the dry crackle of emphysema may at first suggest the presence of pneumonia. But with the aid of the thermometer and consideration of the history and general symptoms there should be no difficulty in forming a correct opinion. The marked alteration in the pulse-respiration ratio—due to the quickening of the breathing out of propoition to the acceleration of the pulse till we may have one breath to every two pulsations instead of one to four—may often guide us to the detection of even a limited pneumonia. Finally, we have to remember the possibility that the pneumonia may be symptomatic of some disease—such as typhoid fever—requiring the fullest attention, or an accident of a tuberculosis which would modify prognosis.

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[6] The Hygienic Prevention of Consumption. Charles Griffin and Co. 1893.

Treatment.—I strongly advocate the stimulant treatment of pneumonia. We have here an illness which is of short duration but often of great severity, and recovery is almost certain if we can keep up the patient's strength till the fever is past. As most of us know by experience, exhaustion may come on so rapidly that if we wait until it shows itself treatment may be too late to counteract it. We may, however, do much to prevent this excessive exhaustion by commencing with stimulant treatment from the first. By stimulant treatment I do not mean necessarily or solely alcoholic treatment, though I consider alcohol one of our most important drugs in the treatment of pneumonia. A full supply of nourishment in suitable form is necessary as well as stimulant drugs, and good nursing is essential. In many cases the nurse is what the practitioner makes her ; and if the medical man shows a knowledge of what good nursing means and gives minute instructions to the nurse, taking measures also to satisfy himself that his instructions are carried out, he will probably have little cause to complain of want of attention on the nurse's part. In severe cases I am in the habit of writing out a time-table for the nurse, stating not only the hour at which nourishment, stimulant, or medicine shall be given, but the nature and quantity of each to be administered on each occasion. I then expect the nurse to keep a diary which I may compare with my time-table at every visit. Whether it is ever allowable to cease giving a patient food, drink, or medicine on the grounds that it can do no good and only bothers the dying patient we need not now discuss. It is certainly never justifiable in a case of pneumonia. I can recall two or three instances of recovery after a patient had been "given up" in which any relaxation of the details of nursing would have taken away the last hope. I cannot too strongly insist on this point.

Local applications over the affected portion of the lung are, in my opinion, very useful. Whether they exert any appreciable influence on the disease may be questioned, but they often ease the patient's discomfort and soothe him. I prefer a poultice as generally most comfortable to the patient, but ice-bags are also recommended. The inhalation of oxygen is often of service, especially when a large extent of lung is consolidated; and I have seen marked improvement in breathing and in general symptoms from placing a patient with high fever in a tepid bath, after which the patient dropped off into a quiet sleep. As to drugs, there is room for individual choice within a certain range, and symptoms will guide us in our selection. I have a liking for carbonâte of ammonia, to which digitalis or strophanthus may be added if there is any danger of cardiac failure. A caution is to be observed as to the use of even simple aperients. Of course the patient must not be allowed to get up, and sudden danger may result from undue action of the bowels. An enema may be needed, and this means of giving both nutriment and alcoholic stimulant also must not be overlooked.

With regard to alcohol, I prefer to begin to use it early in small doses (two to four ounces in the twenty-four hours), and to increase the quantity on the first indication of increasing weakness. Many cases will complete their course without needing any increase of the initial amount, but I have known cases where the alcohol has had to be given in large amount for a day or two. I sometimes tell the nurse in serious cases that if exhaustion is great an extra half ounce of brandy should be given before calling the medical attendant. I well remember, when I was a house physician, a patient with double pneumonia who took about thirty ounces of brandy in one twenty-four hour period. The case was pronounced hopeless by the visiting physician, but I and the nurse decided that we would keep the patient alive over the crisis if it could be done by stimulant, and we succeeded. This experience coming so early in my medical career made a great impression on my mind. I believe there is no better stimulant for use in the exhaustion of pneumonia than alcohol; others I know prefer not to use alcohol at all, but if they are wise they make use of some other stimulant in such cases as the one to which I have just alluded. As I have already intimated, your pneumonic patient will probably recover if you can keep him alive over the tenth day. The personal equation has such a marked influence on the character of the disease that, though we may lay down some general principles for the treatment of pneumonia we must be guided as to details by the individual peculiarities in each case.

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PRINTED AT THE LANCET OFFICE, 423, STRAND. W C.